Environmental Health Perspectives
Vol. 28, pp. 219–222, 1979

Pulmonary and Gastrointestinal Exposure to Cadmium Oxide Dust in a Battery Factory

by E. Adamsson,* M. Piscator,* and K. Nogawa*†

The elimination of cadmium in feces was studied in a group of 15 male workers exposed to cadmium oxide dust in a nickel-cadmium battery factory.

The elimination of cadmium in feces was on the average 619 and 268 μg/day in seven smokers and eight nonsmokers, respectively. The corresponding ranges were 97–2577 and 31–1102 μg/day. The cadmium concentrations in blood were significantly higher in smokers than in nonsmokers, both before and after one month of vacation. Among the smokers there was a significant decrease in the cadmium concentrations during the vacation period, but not among the nonsmokers.

It was estimated that cadmium naturally occurring in food and cigarettes, cadmium excreted from the gastrointestinal tract, and cadmium transported from the lungs by mucociliary clearance to the gastrointestinal tract only could explain up to 100 μg of the cadmium in the feces. Since even among some nonsmokers much higher values for fecal cadmium were recorded, this was interpreted as being the result of ingestion of cadmium from contaminated hands and other body surfaces. Among the smokers, direct oral contact with contaminated cigarettes or pipes is an additional factor; the smokers also inhale cadmium in the tobacco smoke from contaminated cigarettes. Part of that cadmium is transferred to the gastrointestinal tract by mucociliary clearance and also adds to the fecal cadmium.

Introduction

When estimating dose-response relationships for cadmium the methods available today make it possible to get relatively good estimates of the response. The degree of kidney involvement can be estimated by determination of total protein, electrophoretic examination of urine proteins, and the quantitative determination of a specific low molecular weight protein, e.g., β_2-microglobulin (*1*, *2*).

The difficulties with measuring the external dose, i.e., the inhaled amount, have been very large. This is mainly due to the fact that, because of the long biological half-time of cadmium, the lifetime exposure must be evaluated. For estimation of industrial exposure often the present day concentrations in air have been used. It is doubtful if these concentrations accurately reflect environmental conditions in earlier days, since it is obvious that there have been great changes in working conditions over the years. These problems have been discussed by Kjellström (*2*) and by Nordberg (*3*).

Kjellström (*2*) and Adamsson (to be published) investigated the changes over time in the same battery factory described by Friberg (*4*). They found that in this factory the concentrations of cadmium oxide dust in work-room air at present are 100 to 500 times lower than in the 1940's.

In addition to the problem of estimating the external dose, there is also the problem of estimating the absorbed dose, since it is not yet known to what extent the different cadmium compounds are absorbed from the lungs or from the gastrointestinal tract.

Other routes of exposure in addition to inhalation might be of importance for the dose estimate. In 1976 it was shown by Piscator et al. (*5*) that in the above-mentioned battery factory and also in another cadmium-handling factory in Sweden, cigarettes and pipe tobacco were contaminated by

* Department of Environmental Hygiene, Karolinska Institute and the Department of Environmental Hygiene, National Swedish Environment Protection Board, 104 01 Stockholm 60, Sweden.

† Present address: Department of Hygiene, Kanazawa Medical University, Uchinada-machi, Kahoku-gun, Ishikawa-ken 920-02, Japan.

workers, and that that would cause an additional inhalation exposure. This finding prompted further investigations into the problem of contamination and personal hygiene in workers exposed to metal dust. The present study was aimed at finding out to what extent cadmium entered the gastrointestinal tract in these workers, assuming that this could be due both to pulmonary clearance and to direct intake into the mouth via hands and surrounding skin. Since there is exposure not only to cadmium oxide dust, but also to nickel dust in this factory, nickel concentrations in air and feces were determined. The results on the nickel studies are to be reported separately.

Materials and Methods

Fifteen male workers volunteered to participate in the study. They had earlier participated in an 11-week study with serial determinations of cadmium in air, blood and urine in the first quarter of 1977, the results of which will be reported elsewhere. Seven of the workers were smokers. The study was performed during one week in June, 1977. During the first three days of the week, cadmium in air was determined by the use of personal samplers. More details on the method are given elsewhere by Adamsson (to be published). During these three days the workers also recorded on a questionnaire their food and fluid intake and the number of stools. From the third to the fifth day they collected feces in plastic containers. Details have been given by Kjellström et al. (*6*). On the fifth day, whole blood was collected for analysis of cadmium. This fifth day was the last working day before the vacations, and in order to estimate the effect of four weeks without industrial exposure to cadmium in blood levels, new blood samples were taken when the workers returned a month later. On this occasion urine samples were also obtained and urinary cadmium and β_2-microglobulin (β_2-MG) levels were determined. Cadmium in blood and urine was determined with atomic absorption spectrophotometry as described by Kjellström (*2*) and Elinder et al. (*7*). Cadmium in feces was determined as described by Kjellström et al (*6*). β_2-MG was analyzed with radioimmunoassay (*8*) using the Phadebas β_2-MG test (Pharmacia, Uppsala, Sweden). The results are presented with arithmetic means, but since the distributions generally were skewed, statistical tests were made with the logarithms of the values.

Results

According to the questionnaires the dietary habits and the calorie intake seemed to be about the same for all workers. The average fecal weight was 27.4 g

Table 1. Exposure time, cadmium in feces, blood and urine and β_2-microglobulin in urine of workers exposed to cadmium.

Case	Age, years	Working time, years	Cd in air, $\mu g/m^3$ (June)	Fecal Cd (June)		Blood Cd, $\mu g/l$.		Urine Cd, $\mu g/g$ creatinine	β_2-MG, $\mu g/g$ creatinine
				$\mu g/day^a$	$\mu g/g$ dry wt.	June	August	(August)	(August)
Smokers									
1	28	2	9.4	651[b]	27.2[b]	14.5	9.5	1.1	40
2	30	2	15.9	551[b]	18.6[b]	7.3	8.0	1.0	57
3	43	3	9.5	173[b]	8.6[b]	20.2	8.9	1.3	71
4	57	3	13.3	2577	93.9	67.2	62.6	14.7	109
5	58	5	6.2	137[b]	5.9[b]	17.4	9.5	2.5	145
6	48	9	10.1	97	13.1	12.1	11.9	10.1	186
7	58	14	6.5	149	10.3	20.3	15.8	8.0	65
Arithmetic mean	—	—	—	—	—	—	—	—	—
	46	5	10.1	619	25.4	22.7	18.0	5.5	96
Nonsmokers									
8	31	4	4.4	31	3.0	6.2	5.4	2.0	56
9	42	4	6.4	240	10.9	5.0	4.3	1.6	66
10	53	4	7.0	100	7.4	6.2	5.7	2.9	30
11	56	4	6.8	88	2.2	4.9	4.6	0.5	56
12	34	7	5.3	111[c]	4.8[c]	5.2	4.7	1.6	122
13	60	12	8.2	438	11.6	10.4	13.2	9.3	<2[d]
14	46	14	15.8	1102	23.3	7.3	8.3	2.2	17
15	58	26	2.1	32	1.3	10.5	9.2	8.8	510
Arithmetic mean	—	—	—	—	—	—	—	—	—
	48	9	7.0	268	8.1	7.0	6.9	3.6	107

[a] Average of 3 days if not stated otherwise.
[b] Average of 2 stools.
[c] One stool.
[d] pH = 4.98.

dry weight in the nonsmokers as compared to 20.9 g dry weight in the smokers. The average elimination of zinc was 10.5 and 8.7 mg/day, respectively, in nonsmokers and smokers. The fecal concentrations of zinc were, however, the same in both groups, 393 and 398 μg/g dry weight in nonsmokers and smokers, respectively.

In Table 1 it is seen that the cadmium concentrations in air generally were low. They were of the same magnitude as those values found during the long-term sampling period in the spring. The daily elimination of cadmium via feces was, on the average, higher in smokers than in nonsmokers, but the difference was not statistically significant (p = 0.05–0.1). However, when fecal concentrations of cadmium were compared there was a significant difference between smokers and nonsmokers ($p < 0.05$). A significant difference was also found when the cadmium to zinc ratios in feces were compared.

The blood concentrations of cadmium among the nonsmokers were significantly lower than those of the smokers, both before and after the vacation period ($p < 0.01$, $p < 0.05$, respectively). During the vacation period in July there was a significant decrease ($p < 0.01$) in the cadmium blood levels of the smokers, whereas no such change took place among the nonsmokers. The urinary excretion of cadmium was on an average higher among the smokers but the difference was not significant. It should be noted that the nonsmokers had longer employment times than the smokers.

The excretion of β_2-microglobulin in urine was below 200 μg/g creatinine in all workers, except in one man with 26 years of exposure (case 15). This man, however, had the lowest present exposure of the workers.

Among the nonsmokers there was a significant correlation between cadmium in feces and cadmium concentrations in the air (r = 0.88 and 0.86 for daily cadmium elimination and cadmium concentration, respectively). Among the smokers no such significant correlations were found (r = 0.64 and 0.66, respectively).

Discussion

In addition to the exposure via inhalation and pulmonary absorption in industrial environments there may well be an additional exposure from the ingestion of dust. It has long been suspected that oral exposure occurs, and in many industries eating and smoking in work areas have been prohibited. However, hitherto there have been no quantitative data available on the magnitude of this problem. The results from this study show that both smokers and nonsmokers exposed to cadmium oxide dust in a battery factory eliminated large amounts of cadmium via feces. Smokers generally had considerably higher amounts of fecal cadmium than nonsmokers.

To explain the presence of cadmium in feces the following six sources should be considered: (*1*) cadmium occurring naturally in the diet; (*2*) oral exposure to cadmium oxide dust due to surface contamination of food, cigarettes, pipes etc. or transfer from contaminated clothes, hands or other body surfaces; (*3*) cadmium transferred from the lungs by mucociliary clearance to the gastrointestinal tract after inhalation of contaminated air; (*4*) cadmium transferred from the lungs by mucociliary clearance to the gastrointestinal tract after smoking cigarettes containing naturally occurring cadmium; (*5*) cadmium transferred from the lungs by mucociliary clearance to the gastrointestinal tract after smoking cigarettes or pipe tobacco surface-contaminated with cadmium; (*6*) excretion via bile and other parts of the gastrointestinal system.

Of these six sources, (1) and (4) will contribute negligible amounts. In people without occupational exposure to cadmium the daily elimination of cadmium via feces will be about 20 μg, the major part coming from the food and about 2 μg from smoking (*6*). With regard to (6) there are data (*9*) that indicate that the excretion from the gastrointestinal system may be of the same magnitude as excretion via urine, but also this amount will be negligible compared to the large fecal amounts of cadmium found in most cases.

Cadmium oxide dust inhaled from air will be deposited in the respiratory tract and part of the deposited cadmium will be absorbed from the alveoli, whereas the rest will be transferred to a large extent to the gastrointestinal tract via mucociliary clearance from the lung followed by swallowing the mucus containing the cadmium. In the present study all workers were exposed to relatively low concentrations of cadmium in air, 2–15 μg/m^3, and even assuming a daily inhalation of 10 m^3 of air and 50% deposition, amounts of less than 75 μg can be expected to have been transferred to the gut from the lungs.

The total amount in feces from inhalation of air, naturally occurring cadmium in food and cigarettes, and cadmium excreted via the gastrointestinal tract will therefore be no more than 100 μg in this group of workers.

Among the nonsmokers, the cadmium concentrations in air were generally below 10 μg/m^3 on an average, but in several cases relatively large amounts of cadmium were found in feces. The most probable explanation for that finding is that the

workers easily contaminate their clothes and hands and other body surfaces. Even if the air concentrations of cadmium oxide dust are low in the factory, there is always a thin layer of dust on all surfaces in the factory. Depending on personal habits, sweating, etc., the dust might then be brought into the mouth. The dust layer is almost invisible, since even as much as 1 mg Cd will only occupy a volume of 0.1–0.2 mm^3. Among the nonsmokers there was a significant correlation between air levels of cadmium and fecal content of cadmium, indicating that in these work-places the cadmium concentrations in air may also reflect the general dust contamination of the working areas.

The smokers generally had higher fecal amounts of cadmium than the nonsmokers, and in addition to the above-mentioned surface contamination, additional oral exposure may take place when cigarettes or pipes are surface-contaminated with cadmium oxide dust. There will also be increased amounts of cadmium in the inhaled tobacco smoke due to the release of cadmium when the tobacco is burning. It has been reported previously (*5*) that in the same factory cadmium concentrations of up to 25 $\mu g/g$ cigarette and up to several hundred micrograms per gram pipe tobacco were found after they had been handled by the workers. The highest values were recorded in two workers who also took part in the present investigation (cases 4 and 6). It has been estimated that about 45% of cadmium inhaled from tobacco smoke will be absorbed (*10*), and since a large part may be cleared by mucociliary clearance also the gastrointestinal exposure will increase. Furthermore, it should be noted that smoking is not allowed on the working premises and that the workers have been instructed to wash their hands before they start smoking. However, these regulations are often neglected. Furthermore, all smokers carried their cigarettes or pipes in the pockets of their working clothes.

Smokers had significantly higher blood levels of cadmium than nonsmokers. During the vacation there was a significant drop in the blood levels of the smokers, whereas no changes were seen among the nonsmokers. This indicates that part of the high blood cadmium was due to recent exposure caused by the absorption from the lungs of tobacco smoke with high cadmium content. The gastrointestinal absorption of cadmium does not seem to cause such dramatic effects on the blood levels, since among the nonsmokers, even those with relatively large amounts of cadmium in feces did not show a decrease in cadmium levels during the vacation period. About 5% of the dietary cadmium will probably be absorbed (*1*) but it is conceivable that when the cadmium is ingested as particles the absorption will depend on how much of the cadmium oxide dust will go into the solution during its passage.

This study was supported by grant 77/97 from the Swedish Workers' Protection Fund.

REFERENCES

1. Friberg, L., et al. Cadmium in the Environment, 2nd ed., CRC Press, Cleveland, 1974.
2. Kjellström, T. Accumulation and renal effects of cadmium in man. Academic dissertation, Department of Environmental Hygiene, Karolinska Institute, Stockholm, Sweden, 1977.
3. Nordberg, G. F., Ed. Effects and Dose-response Relationships of Toxic Metals. Elsevier, Amsterdam, 1976.
4. Friberg, L. Health hazards in the manufacture of alkaline accumulators with special reference to chronic cadmium poisoning. Acta Med. Scand. 138 (Suppl.): 240 (1950).
5. Piscator, M., Kjellström, T., and Lind, B. Contamination of cigarettes and pipe tobacco by cadmium oxide dust. Lancet II: 587 (1976).
6. Kjellström, T., Borg, K., and Lind, B. Cadmium in feces as an estimation of daily cadmium intake in Sweden. Environ. Res. 15: 242 (1978).
7. Elinder, C.-G., et al. Urinary excretion of cadmium and zinc among persons from Sweden. Environ. Res. 15: 473 (1978).
8. Evrin, P.-E., et al. Radioimmunoassay of β_2-microglobulin in human biological fluids. Scand. J. Clin. Lab. Invest. 28: 439 (1971).
9. Elinder, C.-G., and Pannone, M. Biliary excretion of cadmium. Environ. Health Perspect. 28: 123 (1979).
10. Elinder, C.-G., et al. Cadmium in kidney cortex, liver and pancreas from Swedish autopsies. Arch. Environ. Health 31: 292 (1976).